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Wetwood formation as a host response in white fir

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Abstract

A bacterium associated with wetwood of white fir did not cause wetwood in inoculated trees, and wetwood formed while external water was excluded. Wetwood formed in response to various injuries, suggesting that it is a host response to parenchyma death.

1 Introduction

Wetwood is defined as "a type of heartwood in standing trees which has been internally infused with water" (Ward and Pong 1980). As such, it occupies the internal cylinder of nonliving wood in affected trees. Columns of wetwood, usually continuous with the central cylinder, also occur in association with branch traces (Etheridge and Morin 1962; Lagerberg 1935; Bauch et al. 1975), mechanical wounds (Davidson et al. 1959; Campbell and Davidson 1941; Hornibrook 1950), insect attacks (Johnson and Shea 1963; Wickman and Scharpf 1972; Owen and Wilcox 1982), and root and butt decay (Coutts and Rishbeth 1977; Lagerberg 1935; Linzon 1958; Schmitz and Jackson 1927). Such tissues are also nonliving. Thus, wetwood in such tissues, which in some cases has been called "pathological wetwood" (Bauch et al. 1978, 1979) or "wet pathological heartwood" (Coutts and Rishbeth 1977), may be homologous with what is termed "protection wood" (Hepting and Blaisdell 1936; Jorgensen 1962), "reaction wood" (Shain 1967, 1971), "pathological heartwood" (McNabb et al. 1959) or "discolored sapwood" (Hart and Johnson 1970).

As discussed in recent reviews (Hartley et al. 1961; Ward and Pong 1980), various theories are espoused by different authors as to the cause of wetwood. The association of wetwood with branch stubs and wounds has led to the conclusion that external water enters through such pathways and accumulates in wetwood (Etheridge and Morin 1962; Lagerberg 1935) and to the proposal that wetwood is a disease associated with bacteria entering through the same pathways (Carter 1945; Seliskar 1952; Murdoch and Campana 1981). Although inoculations have been claimed to demonstrate a causal role for bacteria in wetwood (Carter 1945; Day 1924; Dowson 1937; Crandall 1943; Seliskar 1952), careful examination of such data and the limited number of tree species examined suggest

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that generalizations and assumptions of bacterial etiology of wetwood, at least in fir (*Abies* spp.) (Brill et al. 1981; Ulrich 1981), may be unfounded. Data of Coutts and Rishbeth (1977) suggest that wetwood in *A. grandis* may form in the absence of bacteria or external water.

In white fir (*A. concolor*) wetwood is formed in association with injuries, branch stubs, and wood decay as well as in the central cylinder of heartwood. A coryneform bacterium (WWB) has been consistently isolated in numbers as high as 10^6 per ml wetwood fluid and has been characterized by Wilcox and Oldham (1971, 1972). The studies reported here were designed to examine the role of wounds, *Heterobasidion annosum* infection, WWB, and ray parenchyma death in formation of wetwood in fir.

2 Materials and methods

2.1 Field observations

Patterns of wetwood development in naturally wounded and/or diseased trees were observed in various stands of the Lassen, Modoc and Stanislaus National Forests in the northern Sierra Nevada in California. Freshly cut stumps left by logging crews, trees uprooted in randomly selected plots in conjunction with another study, and trees felled in and adjacent to *Heterobasidion annosum* (Fr.) Brefeld (*Fomes annosus* [Fr.] Cooke) infection centers provided material for examination.

2.2 Induction of wetwood in trees

Wetwood response to various treatments was studied in trees in the Stanislaus National Forest. White firs with average d.b.h. of 14 cm were selected for extremes of growth rate based on morphological characters, including distance between internodes on the terminal, crown form, and smoothness of bark. As a result, ages were later found to vary from 17 to 129 years. Five treatments were systematically assigned three per tree to 10 fast-growing (mean radial growth rate = 5 mm/yr) and 10 slow-growing (mean radial growth rate = 1.4 mm/yr) trees, and one tree of each class received each treatment individually, giving a total of 30 trees. The treatments were: a. 0.01 M mercuric chloride was applied to test the effect of slow killing of the parenchyma (Coutts and Rishbeth 1977); b. a freshly prepared aqueous turbid suspension of the WWB (Wilcox and Oldham 1971, 1972; kindly supplied by W. W. Wilcox, Univ. of Calif. Forest Products Laboratory, Richmond, CA, USA) was applied to assess its ability to cause wetwood; c. 0.1 M KCl ($\psi_\pi = -5$ bars) was included to test the effect of an osmoticum similar to that in wetwood (Worrall and Parmeter 1982); d. sterile distilled water; and e. blank (no treatment). At 1.5 m above soil line, we shaved the bark smooth if it was rough, swabbed it and the drill bit with ethanol, and drilled 1 cm diameter holes angling down 20° from the horizontal to a depth of one-third tree diameter. Ten ml test solution was introduced, and holes were sealed with polyethylene sheeting and tree seal. After 3 months, 1 m stem lengths centered at the treatment were dissected by serial cross-section to determine vertical extent of wetwood and drying.

2.3 Wetwood formation in wounded trees

A second experiment in natural stands was designed to examine wetwood formation in response to wounds similar to those commonly inflicted on fir during logging. Wounds (10 cm wide × 30 cm with lower edge 20 cm above soil line) were made by excising bark and scoring heavily with an axe on opposite sides of trees averaging 18 cm d.b.h. One wound per tree was sprayed with 3 ml conidial suspension of *Heterobasidion annosum*.

2.4 Effect of bacteria on wetwood and colonization by Heterobasidion annosum

A third experiment was designed to examine wetwood formation and *H. annosum* development in co-inoculations with the WWB. Bacteria were washed from 15-day-old YDCP plates with 0.1 M phosphate buffer (pH 6.4), washed twice by centrifugation and resuspension, then suspended in buffer at approximately 10^6 cells/ml. Bacterial suspension or sterile buffer was vacuum infiltrated into sterile dowels which had been split lengthwise into quarter sections (0.6×6 cm).

Heterobasidion annosum was incubated with similar sections for 3 months. Holes were drilled 1.5 m above soil line on opposite sides of white firs, 13–20 cm d.b.h., on the Stanislaus N.F. Each hole received a dowel section infested with *H. annosum* and a second dowel with WWB inoculum or buffer control.

2.5 Wetwood formation in inoculated seedlings

A fourth experiment was performed with 2-year-old fir seedlings in the greenhouse inoculated with isolates of *H. annosum* from pine or fir. Detailed methods are described elsewhere (WORRALL et al. in press). Briefly, incisions were made with sterile technique just above soil line, infested wedges or sterile control wedges were inserted, and the inoculation area was tightly wrapped with waterproof thermoplastic film.

3 Results

3.1 Field observations

Wetwood generally occurred as a clearly differentiated cylinder in the inner wood at the butt of sectioned trees. Occasionally only the outer heartwood was occupied by wetwood. In most cases a dry transition zone, 1–5 rings in width, was distinctly visible, especially on shaved sections. Apparent wetness, color, odor and the drier transition zone typical of wetwood were less pronounced in the upper bole. Lobes of wetwood extended from the central cylinder above and below branch traces, tapering to a point at their horizontal extension (Fig. 1A).

Such lobes also occurred in association with mechanical wounds and attacks by the bark beetle *Scolytus ventralis* (Coleoptera: Scolytidae), but terminated broadly where they met such lesions. Small lesions occasionally had wedges of wetwood internal to them which were unconnected with the central cylinder and were surrounded by normal sapwood.

Heterobasidion annosum caused decay columns which were wide and extensive at the root collar and tapered to incipient decay above the soil level. Such decayed areas were typically horseshoe-shaped and roughly occupied the transition zone (Fig. 1B). Wetwood was usually present in the wood external and internal to the decay, bulging outward in contrast to the undecayed side of the stem.

3.2 Induction of wetwood in trees

The treatment solutions tested for wetwood induction caused the formation of dry zones above and below the wound, within which wetwood columns were found in some cases (Fig. 1C, 2). Replications of the blank treatment were to few to analyze owing to interference from previous wounds or mistletoe infections. Large variation in lengths of dry zones and wetwood between trees was apparent. There was no significant difference between fast-growing and slow-growing trees for any treatment ($P = 0.05$) so they were combined for further analysis. The bacterial treatment resulted in less wetwood than the water control though this was not significant. Attempts to reisolate the bacterium failed.

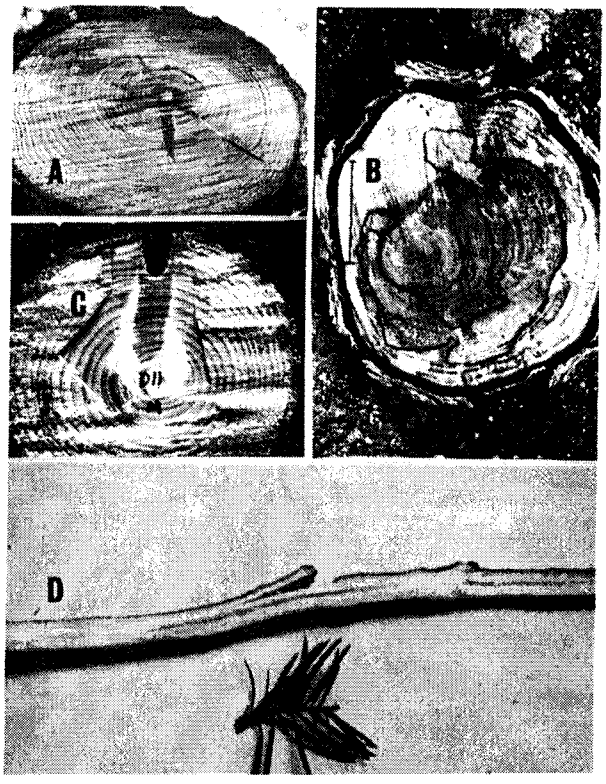

Fig. 1. Wetwood and decay encountered in observations and experiments on wetwood formation in white fir. A. Wetwood in a healthy tree showing lobes associated with branch traces. B. Decay caused by *Heterobasidion annosum* and associated wetwood in a tree recently killed by the fungus. Obvious decay is outlined; wood internal to this is undecayed wetwood. C. Wetwood formed within a dry zone resulting from treatment with $HgCl_2$. No natural heartwood was present in this tree. D. Streak of wetwood in a seedling inoculated with *H. annosum*

The KCl treatment also was not different from controls. Only $HgCl_2$ induced significantly longer dry zones ($P = 0.05$) and wetwood ($P = 0.1$) although several trees showed essentially no response. In view of the large variation between trees and the lack of pairing of controls and treatments on the same tree in this experimental design, it is felt that $P = 0.1$ is a reasonable level at which to reject the null hypothesis (mean length of wetwood for controls = mean length of wetwood for $HgCl_2$).

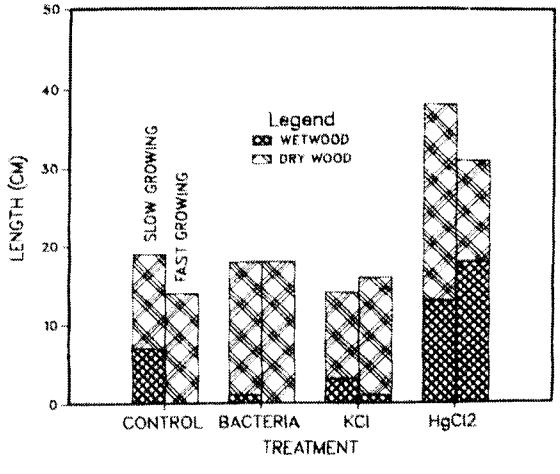

Fig. 2. Mean length (cm) of dry zones (DW) and wetwood (WW) formed in slow and fast growing white firs in 3 months in response to various treatments. Treatments induced drying of columns of wood within which wetwood formed to varying extents. Means for slow and fast growing trees are not significantly different. When growth rate classes were combined, only $HgCl_2$ resulted in significantly greater mean length of DW ($P = 0.05$) and WW ($P = 0.1$) than water controls

3.3 Wetwood formation in wounded trees

After three years, considerable wetwood was found in response to mechanical wounding with and without inoculation of *H. annosum* (Table 1). Of 9 trees examined, *H. annosum* was found only in two inoculation treatments and was also recovered from two uninoculated wounds. The effect of wounding was generally seen as a lobe of wetwood extending from and continuous with the central wetwood. This lobe extended almost to the surface at the wound site and was shorter radially in sections farther up and down from the wound. Great variation was observed among trees in length, apparent wetness and color of wetwood columns. Although in many trees inoculation was apparently unsuccessful and wetwood formation on both sides was equal, a paired t-test indicated that wetwood columns overall were significantly longer on inoculated wounds (P = 0.05).

Table 1

Length of wetwood development and Heterobasidion annosum colonization (cm) in trees which were mechanically wounded with and without spray-inoculation of H. annosum conidia

	Length of wetwood column[1]		Length of *H. annosum* colonization[1]	
	Inoculated	Uninoculated	Inoculated	Uninoculated
	100	0	0	0
	80	80	0	0
	120	120	0	80
	140	60	20	0
	100	40	0	0
	100	100	0	0
	0	0	120[2]	
	140	140	20	0
	40	0	0	0
$\bar{x}$[3] =	91	60		

[1] Sampled at 20 cm increments.
[2] *Heterobasidion annosum* extensively colonized the heartwood of this tree on both sides.
[3] Means for length of wetwood in response to inoculated and uninoculated wounds are significantly different (P = 0.05) with a paired t-test.

Table 2

Length[1] (cm) of wetwood development, Heterobasidion annosum colonization, and decay in six trees inoculated with H. annosum with (+) and without (−) the wetwood bacterium (WWB)

Means for +WWB and −WWB are not significantly different

Length of wetwood effect		Length of *H. annosum* colonization		Length of decay	
+WWB	−WWB	+WWB	−WWB	+WWB	−WWB
80	100	200[2]		20	60
200	160	0	0	0	0
80	60	80	–	40	20
120	100	140	120	100	80
180	140	180	60	100	100
100	100	180[2]		20	40

[1] Sampled at 20 cm increments.
[2] *H. annosum* extensively colonized the heartwood on both sides.

3.4 Effect of bacteria on wetwood and H. annosum development

No significant difference in wetwood formation or decay in response to *H. annosum* inoculation with and without bacteria was observed after 3 years (Table 2). *Heterobasidion annosum* extensively colonized several trees, making it difficult to determine with which treatment it was associated, and conidiophore production on some discs was quite sparse. All remaining trees were colonized except one which was relatively fast growing and had wetter, darker colored wetwood and greater vertical extent of wetwood formation than the other trees.

3.5 Wetwood formation in inoculated seedlings

Inoculation of fir seedlings with 18 isolates of *H. annosum* resulted in 0–61 % mortality with an average of 21 % (experiment 2 of WORRALL et al., in press). Wetwood was not detected in dead seedlings or in controls, none of which died. Of the remaining seedlings which were considered healthy on the basis of external symptoms, 27 % had wetwood associated with the inoculation (Table 3, Fig. 1D). The term was applied on the basis of the following characters: a wet appearance (verified by observing drops of moisture expressed with a scalpel point), a dry transition zone separating the tissue from unaffected sapwood, brown color, and presence of dark amorphous deposits in the parenchyma together with a greatly reduced content of starch. Seedling stems were approximately 8–12 mm thick and the wetwood was generally several mm thick and 1–4 cm long. As in the field experiments, individual variation in wetwood formation was apparent: some seedlings showed no res-

Table 3

Formation of wetwood in fir seedlings which survived inoculation with isolates of Heterobasidion annosum from pine or fir and which were not colonized, colonized only at the point of inoculation or colonized beyond the point of inoculation

Isolates from pine	Fa[-][1]	Fa[+2]	Fa[+3]	Total percent
Las 8P	1/6[4]	1/7	2/8	19
Bog 3P	3/11	6/12	1/1	42
Mod 12P	1/7	3/6	2/4	35
Las 11P	0/4	6/12	0/1	35
JL1	3/14	0/9	0/1	13
PP1	2/5	5/13	2/6	38
JP6	0/13	3/13	0/2	11
Las 3P	0/4	2/14	2/5	17
Bog 7P	6/26	0/1	0/0	22
Total percent	18	30	32	25
Isolates from fir	**Fa[-][1]**	**Fa[+2]**	**Fa[+3]**	**Total percent**
Las 7F	0/1	1/1	3/5	57
Las 1F	0/1	0/1	2/14	13
Mod 15F	0/0	0/1	4/9	40
WF5	1/4	4/4	5/16	42
Las 21F	4/22	1/2	0/1	20
WF3	0/1	2/2	2/7	40
WF1	4/11	3/10	3/6	37
Las 17F	4/28	0/0	0/0	14
Mod 14F	8/14	1/8	0/4	35
Total percent	26	41	31	30

[1] *H. annosum* not detected in seedling.
[2] *H. annosum* colonized only tissue immediately adjacent to inoculum.
[3] *H. annosum* colonized seedling beyond point of inoculation.
[4] Number of seedling with wetwood/n.

ponse, some showed only regions of necrotic parenchyma which gave the wood a grey color, and others showed the above wetwood symptoms over varying portions of their length.

In general the isolates from fir caused greater percentage of wetwood than those from pine, but these differences were not significant, nor was there a difference between seedlings colonized beyond the point of inoculation and those colonized only immediately around it. Data for the two isolate groups and for the two categories of colonization were combined and X^2 analysis showed significantly more frequent wetwood formation in colonized seedlings than in those in which the fungus was not apparent.

4 Discussion

These results demonstrate that neither bacteria nor external water is necessary for production of wetwood in white fir. Rather, evidence suggests that wetwood formation is a response to parenchyma death which may result from wounding or fungal attack as well as normal heartwood formation.

The WWB consistently present in wetwood in numbers as high as 10^6 colony forming units/ml showed no effect on wetwood formation when inoculated into sapwood. Control inoculations with sterile water induced as much or more wetwood than the WWB. The WWB also had no effect on wetwood formation when co-inoculated with *H. annosum*. In agreement with results of COUTTS and RISHBETH (1977), $HgCl_2$ induced more wetwood than controls. It seems unlikely that bacteria would grow in concentrations of mercury high enough to kill host parenchyma. In seedling inoculations, wetwood formed in response to *H. annosum* but not control inoculations. If bacteria were responsible, they presumably would have equal chances of invading either control or fungal inoculations. These data do not preclude the possibility that another bacterium is capable of inducing wetwood. However, it seems clear that wetwood is a non-specific response which can occur in the absence of bacteria.

Penetration and accumulation of external water also seems unlikely for several reasons. Like COUTTS and RISHBETH (1977) we obtained wetwood formation in response to treatments even when excluding water in field inoculations with polyethylene. In the seedling experiments no branch stubs were present, the wound was wrapped tightly with waterproof film, and seedlings were watered carefully to avoid wetting the inoculum, yet wetwood occurred in response to inoculation.

The concept of wetwood as a host response to death of ray parenchyma is suggested by its occurrence in the same anatomical regions where heartwood, reaction wood or pathological heartwood occur in species lacking wetwood. The formation of wetwood in tissue killed by $HgCl_2$ supports this concept. Wetwood formation in the central cylinder of heartwood occurs as a normal part of a series of changes preceding and during cell death, and involves accumulation of dissolved solutes (COUTTS and RISHBETH 1977; WORRALL and PARMETER 1982). Wetwood was formed in large trees in response to wounds and formed to a greater extent in trees inoculated with *H. annosum*. In seedlings, wetwood formed only in response to the fungus. Apparently the degree of wetwood formation is determined in part by the amount of parenchyma killed. While the small sterile wounds made during control seedling inoculations were relatively nontraumatic, inoculation with *H. annosum* and the wounds in the field experiments, large and open to microbial challenge, resulted in sufficient cell death for recognizable wetwood formation.

Potassium chloride was originally included in the wetwood induction experiment to explore the osmotic hypothesis for wetwood formation (COUTTS and RISHBETH 1977; WORRALL and PARMETER 1982). The wetwood osmoticum exists in a static fluid in the presence of other features necessary for wetwood formation. One of these is the dry transition zone separating wetwood from sapwood and bridged by living parenchyma

(WORRALL and PARMETER 1982). This dry zone may function as a semipermeable membrane, allowing solute transport to the wetwood border via living parenchyma, while preventing liquid flow across the dry transition zone. Water, responding to the osmotic imbalance (COUTTS and RISHBETH 1977; WORRALL and PARMETER 1982), may enter the sapwood through the parenchyma or across the tracheids as a vapor. These conditions were not met in the KCl experiment, where the salt was introduced into and probably diluted by the transpiration stream with no intervening dry zone as in natural wetwood.

It has been suggested that low oxygen availability and inhibition by organic acids in wetwood may function to slow the progress of heart-rot (WORRALL and PARMETER, manuscript in preparation; VAN DER KAMP et al. 1979). *Echinodontium tinctorium*, which causes heart-rot in the upper bole of firs, normally does not invade wetwood (AHO 1974), and decay is reportedly rare also in wetwood of elm (MURDOCH and CAMPANA 1981). Balsam fir heartwood is known to have heat-sensitive anti-fungal properties (ETHERIDGE 1962). The preferential colonization by *H. annosum* of the wood in the transition zone suggests that both sapwood and wetwood are more resistant than this zone. The sapwood may be capable of active resistance while the nonliving wetwood may possess passive resistance analogous to that of resinous and extractive-rich heartwood of other species. Apparently any such inhibition of *H. annosum* is not a direct effect of WWB since growth and decay *in vivo* was unaffected by co-inoculation with WWB. Similarly, WWB grown for 2 weeks under aerobic or anaerobic conditions on YDCP showed no inhibitory activity when *H. annosum* was plated beside it (unpublished).

The large tree-to-tree variation in quantity and quality of wetwood production observed in these experiments is of interest with regard to its proposed function in resistance to heart-rot. In the WWB co-inoculations, *H. annosum* failed to colonize and decay only the tree which responded with the greatest length and degree of wetwood. In contrast, the tree which did not form wetwood in the wounding study was colonized far more extensively than those that did. Seedling inoculations showed that wetwood was not formed in seedlings killed by the fungus but was often present in apparently more resistant individuals. Our data do not indicate that length of wetwood response is greater in faster-growing trees, and previous surveys showed no correlation between growth rate and percent of cross-sectional area occupied by natural wetwood (WORRALL and PARMETER 1982). However, there is some indication that tree vigor has an important role in wetwood formation (COUTTS and RISHBETH 1977). Our data do not exclude the possibility that faster-growing trees form wetwood more quickly in response to injury or that they form a qualitatively different wetwood.

These results indicate that wetwood may form in the absence of WWB or external water. Although bacteria probably contribute to secondary characteristics such as low oxygen availability, organic acid production, methanogenesis and pectolytic activity (VAN DER KAMP et al. 1979; SCHINK et al. 1981; WORRALL and PARMETER 1982; ZEIKUS and WARD 1974), they are apparently not necessary to its formation. These results and others (COUTTS and RISHBETH 1977; WORRALL and PARMETER 1982) suggest the view that wetwood is a host response to sapwood death. Thus, anomalous wetwood observed in addition to the normal central cylinder may be a symptom of disease or injury rather than a disease as such.

Summary

Wetwood in white fir (*Abies concolor*) occupies the central cylinder of heartwood and is also associated with branch gaps, wounds, insect scars, and root and butt decay. In two experiments, a bacterium associated with wetwood did not cause wetwood, and wetwood formed while external water was excluded. Wetwood formation resulted from large wounds, treatment with $HgCl_2$, and inoculation with *Heterobasidion annosum*. Wetwood formed in seedlings which survived inoculation with *H. annosum* but not in those which were killed by the fungus or which received control inoculations. Large variation was observed among trees in quantity or quality of wetwood formed in response to various treatments.

Observations of natural columns and circumstantial evidence from inoculations indicate that wetwood may be inhibitory to *H. annosum*. Results suggest that wetwood is a host response to parenchyma death rather than a baterial disease or accumulation of external water.

Résumé

Formation de bois humide comme moyen de défense du Sapin concolor

Le bois humide chez le sapin (*Abies concolor*) occupe le cylindre central du bois de coeur et s'associe également avec les bris de branches, blessures, scarifications dues aux insectes et pourritures de racines et du collet.

Au cours de deux expériences, une bactérie associée avec le bois humide n'a pas reproduit le symptôme et du bois humide formé malgré l'absence d'eau extérieure. La formation de bois humide résulte de blessures importantes, du traitement au $HgCl_2$ et d'inoculation avec *Heterobasidion annosum*. Le bois humide s'est formé chez les semis qui ont survécu à une inoculation avec *H. annosum*, mais non chez ceux qui ont été tués par le champignon ou qui ont reçu des inoculations témoins. De larges variations ont été observées entre arbres quant à la quantité et à la qualité du bois humide formé en réponse à divers traitements. Des observations de colonnes de pourriture naturelle et des faits d'évidence selon les cas à partir d'inoculations montrent que le bois humide peut être inhibiteur vis-à-vis de *H. annosum*. Ces résultats laissent supposer que le bois humide serait une réponse de l'hôte à la mort du parenchyme plutôt qu'une maladie bactérienne ou une accumulation d'eau venant de l'extérieur.

Zusammenfassung

Naßkernbildung als Abwehrreaktion der Tanne

Bei *Abies concolor* nimmt der Naßkern den zentralen Kernholzzylinder ein. Er ist außerdem mit Astlöchern, Wunden, Insektengängen sowie mit Wurzel- und Stockfäule verbunden. In zwei Versuchsanstellungen war ein im Naßkern lebendes Bakterium nicht zur Auslösung des Naßkerns befähigt. Andererseits bildete sich der Naßkern auch, wenn äußeres Wasser ferngehalten wurde. Die Naßkernbildung ging aus von großen Wunden, von $HgCl_2$-Behandlung und von *Heterobasidion annosum*-Inokulationen. Naßkern bildete sich auch in Pflanzen, die eine *Heterobasidion*-Infektion überstanden, nicht aber in solchen, die von diesem Pilz getötet wurden oder an denen Kontroll-Inokulationen stattgefunden hatten. Zwischen verschiedenen Bäumen wurde eine erhebliche Schwankungsbreite in der Art und Intensität der Naßkernbildung in Abhängigkeit von verschiedenen Behandlungen festgestellt. Beobachtungen an natürlichen Naßkernen und entsprechende Bilder nach Inokulationen lassen erwarten, daß Naßkern die Ausbreitung von *H. annosum* hemmt. Die Ergebnisse deuten an, daß man den Naßkern eher als eine Reaktion auf das Absterben von Parenchymzellen werten kann, als daß er eine Bakterienkrankheit oder eine Akkumulation externen Wassers darstellt.

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